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=> s endoglin or Alk-2 or activin receptor like kinase or elastin or Eph4B or EphrinB2
L1 18430 ENDOGLIN OR ALK-2 OR ACTIVIN RECEPTOR LIKE KINASE OR ELASTIN OR EPH4B OR EPHRINB2

=> s endothel? 5(a) remodel?
L2 0 ENDOTHEL? 5(A) REMODEL?

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=> s endothel? 5(a) re-model?
L3 0 ENDOTHEL? 5(A) RE-MODEL?

=> s endothel? 3(s) remodel?
L4 6 ENDOTHEL? 3(S) REMODEL?

=> s l1 AND L4
L5 0 L1 AND L4

=> dup rem l4
PROCESSING COMPLETED FOR L4
L6 4 DUP REM L4 (2 DUPLICATES REMOVED)

=> d bib abs 1-
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CONTINUE? Y(N):y

L6 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2002 ACS
AN 1998:153517 CAPLUS
DN 128:293474
TI Vascular remodeling and growth factor gene expression in the rat lung during hypoxia
AU Pfeifer, Michael; Blumberg, Friedrich C.; Wolf, Konrad; Sandner, Peter; Eisner, Dietmar; Riegger, G. unther A. J.; Kurtz, Armin
CS Klinik und Poliklinik fur Innere Medizin II, University of Regensburg, Franz-Josef-Strauss-Allee 11, Regensburg, D-93042, Germany
SO Respiration Physiology (1998), 111(2), 201-212
CODEN: RSPYAK; ISSN: 0034-5687
PB Elsevier Science B.V.
DT Journal
LA English
AB Recent studies suggest that the vasoactive peptides endothelin-1 and -3 and the mitogens VEGF and PDGF-A and -B could be involved in the pathogenesis of hypoxic pulmonary hypertension. Whether these peptides could also be involved in the vascular remodeling occurring during chronic hypoxia (10% oxygen; 1 and 3 wk) was investigated in the rat. Hypoxia increased systolic right ventricular pressure and typical morphol. signs of vascular remodeling were found. This was accompanied by increased ET-1 and the ET-3 mRNA expression after acute (6 h) and chronic hypoxia of 1 and 3 wk. In contrast, no effects of hypoxia on the gene expression of VEGF and PDGF-A and -B in the lung were found. Thus, ET-3 in addn. to ET-1 could be involved in the process of hypoxia-induced vascular remodeling, whereas it appears less likely that the mitogens VEGF and PDGF-A and -B are essentially involved in the pathogenesis of hypoxic pulmonary hypertension.

L6 ANSWER 2 OF 4 EMBASE COPYRIGHT 2002 ELSEVIER
SCI. B.V.DUPLICATE 1
AN 1998056433 EMBASE
TI Carvedilol update IV: Prevention of oxidative stress, cardiac remodeling and progression of congestive heart failure.
AU Feuerstein G.Z.; Shusterman N.H.; Ruffolo R.R. Jr.
SO Drugs of Today, (1998) 34/SUPPL. B (1-23).
Refs: 93

ISSN: 0025-7656 CODEN: MDACAP
 CY Spain
 DT Journal; General Review
 FS 018 Cardiovascular Diseases and Cardiovascular Surgery
 030 Pharmacology
 037 Drug Literature Index
 LA English
 SL English
 AB On May 29, 1997, the United States Food and Drug Administration granted final approval for the use of carvedilol in the treatment of mild to moderate congestive heart failure; in this action, the United States joined 20 countries worldwide that have approved carvedilol (Coreg.RTM./Kredex.RTM.) for treatment of hypertension and congestive heart failure. Carvedilol is also approved for the treatment of angina in several countries. Carvedilol (Fig. 1) is a chemically distinct and pharmacologically unique agent that possesses multiple pharmacological actions, including: 1) nonselective .beta.-adrenoceptor blockade, 2) .alpha.1-adrenoceptor blockade, 3) potent antioxidant activity, and 4) regulation of genes involved in cardiovascular organ ***remodeling*** and apoptosis. Based on this pharmacological profile, carvedilol is uniquely positioned to inhibit several of the major pathological processes that drive the progression of congestive heart failure, including: 1) hemodynamics: reduction of preload, afterload and heart rate; 2) neurohormonal: inhibition of the sympathetic nervous system, renin-angiotensin system and ***endothelin*** ; ***3***) oxidative stress: scavenging potentially toxic oxygen radicals and restoring endogenous antioxidants; 4) genomic reformatting: suppression of several genes associated with pathological organ ***remodeling*** . Thus, carvedilol, through its multiple actions, has the capacity to provide broad cardiovascular organ protection. As a result of these multiple actions, carvedilol, when used in conjunction with standard therapy for heart failure (i.e., diuretics, digoxin, and angiotensin-converting enzyme inhibitors), significantly reduced morbidity, mortality and hospitalization in patients with congestive heart failure of either ischemic or nonischemic (i.e., idiopathic dilated cardiomyopathy) origin, independent of disease severity (mild to moderate) or left ventricular function (ejection fraction). The highly favorable clinical outcomes from the large multicenter clinical trials conducted with carvedilol in the United States and Australia/New Zealand merits a detailed update of the unique mechanisms of action of carvedilol, and a thorough review of the clinical trial results. Accordingly, we will highlight in this update our previous experimental findings with carvedilol as well as more recent data that shed light on the mechanisms by which this drug produces its effects in congestive heart failure. In addition, an update of the results from the large multicenter clinical trials, which formed the basis for the approval of the drug for the treatment of heart failure, will be presented.

L6 ANSWER 3 OF 4 EMBASE COPYRIGHT 2002 ELSEVIER
 SCI. B.V.DUPLICATE 2
 AN 97289277 EMBASE
 DN 1997289277
 TI Carvedilol update iv: Prevention of oxidative stress, cardiac remodeling and progression of congestive heart failure.
 AU Feuerstein G.Z.; Shusterman N.H.; Ruffolo R.R.
 CS R.R. Ruffolo, Pharmacological Sciences, SmithKline Beecham Pharmaceuticals, UW2523, 709 Swedeland Road, King of Prussia, PA 1904-0939, United States
 SO Drugs of Today, (1997) 33/7 (453-473).
 Refs: 93
 ISSN: 0025-7656 CODEN: MDACAP
 CY Spain
 DT Journal; General Review
 FS 006 Internal Medicine

018 Cardiovascular Diseases and Cardiovascular Surgery
 030 Pharmacology
 037 Drug Literature Index
 038 Adverse Reactions Titles
 LA English
 SL English
 AB Summary On May 29, 1997, the United States Food and Drug Administration granted final approval for the use of carvedilol in the treatment of mild to moderate congestive heart failure. In this action, the United States joined 20 countries worldwide that have approved carvedilol (Coreg.RTM./Kredex.RTM.) for treatment of hypertension and congestive heart failure. Carvedilol is also approved for the treatment of angina in several countries. Carvedilol (Fig. 1) is a chemically distinct and pharmacologically unique agent that possesses multiple pharmacological actions, including: 1) nonselective .beta.-adrenoceptor blockade, 2) .alpha.1-adrenoceptor blockade, 3) potent antioxidant activity, and 4) regulation of genes involved in cardiovascular organ ***remodeling*** and apoptosis. Based on this pharmacological profile, carvedilol is uniquely positioned to inhibit several of the major pathological processes that drive the progression of congestive heart failure, including: 1) hemodynamics: reduction of preload, afterload and heart rate; 2) neurohormonal: inhibition of the sympathetic nervous system, renin-angiotensin system and ***endothelin*** ; ***3***) oxidative stress: scavenging potentially toxic oxygen radicals and restoring endogenous antioxidants; 4) genomic reformatting: suppression of several genes associated with pathological organ ***remodeling*** . Thus, carvedilol, through its multiple actions, has the capacity to provide broad cardiovascular organ protection. As a result of these multiple actions, carvedilol, when used in conjunction with standard therapy for heart failure (i.e., diuretics, digoxin, and angiotensin-converting enzyme inhibitors), significantly reduced morbidity, mortality and hospitalization in patients with congestive heart failure of either ischemic or nonischemic (i.e., idiopathic dilated cardiomyopathy) origin, independent of disease severity (mild to moderate) or left ventricular function (ejection fraction). The highly favorable clinical outcomes from the large multicenter clinical trials conducted with carvedilol in the United States and Australia/New Zealand merits a detailed update of the unique mechanisms of action of carvedilol, and a thorough review of the clinical trial results. Accordingly, we will highlight in this update our previous experimental findings with carvedilol as well as more recent data that shed light on the mechanisms by which this drug produces its effects in congestive heart failure. In addition, an update of the results from the large multicenter clinical trials, which formed the basis for the approval of the drug for the treatment of heart failure, will be presented.

L6 ANSWER 4 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
 AN 1993:475290 BIOSIS
 DN PREV199396108890
 TI Endothelin-1 and endothelin-3 induce chemotaxis and replication of pulmonary artery fibroblasts.
 AU Peacock, Andrew J. (1); Dawes, Keith D.; Shock, Anthony; Gray, Andrew J.; Reeves, John T.; Laurent, Geoffrey J.
 CS (1) Pulmonary Vascular Unit, Dep. Respiratory Med., Western Infirmary, Glasgow G11 6NT UK
 SO American Journal of Respiratory Cell and Molecular Biology, (1992) Vol. 7, No. 5, pp. 492-499.
 ISSN: 1044-1549.
 DT Article
 LA English

AB The ***remodeling*** of pulmonary vessels that occurs in association with pulmonary hypertension involves, in part, thickening of the adventitia. The stimulus for this process is not understood. One explanation is that endothelial cells secrete a growth factor that expands the local population of fibroblasts by acting as a chemoattractant and mitogen. Endothelins are a family of potent newly discovered vasoactive peptides. One of these compounds, endothelin-1 (ET-1), is secreted by endothelial cells and is known to constrict pulmonary vessels. Another, ***endothelin*** - ***3*** (ET-3), is not secreted by endothelial cells and is less potent as a pulmonary vasoconstrictor. We hypothesized that the endothelins may have the capacity both to constrict these vessels and to initiate fibroblast chemotaxis and replication. Here we investigated the effects of both ET-1 and ET-3 on the chemotaxis and replication of fibroblasts derived from pulmonary vessels. Cells were isolated from rat pulmonary arteries, cultured in medium and 10% newborn calf serum, and used between passages 2 and 5. Chemotaxis was assessed using a modified Boyden chamber with a polycarbonate filter (pore size, 8 μ m) separating cells in the upper chambers from endothelin in the lower chambers. Replication was assessed both by direct cell counts and by a colorimetric assay based on uptake and subsequent release of methylene blue. Both ET-1 and ET-3 induced chemotaxis of pulmonary artery fibroblasts and did so in a dose-dependent fashion. The maxima for both peptides occurred at a concentration of about 10⁻⁷ M, when chemotaxis was greatest for ET-1 (22 \pm 1.4 versus 14 \pm 1.8 cells/grid (mean \pm SEM), P < 0.05). Both endothelins also induced fibroblast replication, but once again the effect of ET-1 was greater. Maximal mitogenic response occurred at a concentration of about 10⁻⁵ M and was 35 \pm 3.1% above control for ET-1 and 20 \pm 1% above control for ET-3. These results were obtained in the presence of 2% newborn calf serum, but results were very similar in the absence of serum. This study suggests that the endothelins, known to be vasoconstrictors, also have the capacity to attract fibroblasts and stimulate their replication. This may have important implications in pulmonary hypertension, suggesting a possible link between pulmonary vasoconstriction and an important component of vascular ***remodeling***.

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 L1 18430 S ENDOGLIN OR ALK-2 OR ACTIVIN RECEPTOR LIKE KINASE OR ELASTIN
 L2 0 S ENDOTHEL? 5(A) REMODEL?
 L3 0 S ENDOTHEL? 5(A) RE-MODEL?
 L4 6 S ENDOTHEL? 3(S) REMODEL?
 L5 0 S L1 AND L4
 L6 4 DUP REM L4 (2 DUPLICATES REMOVED)

=> s l1 5(s) remodel?

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=> s remodel? 5(s) l1

L7 0 REMODEL? 5(S) L1

=> s l1 and remodel?

L8 542 L1 AND REMODEL?

=> d l8 and vector?

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L8 ANSWER 1 OF 542 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
 AN 2002:372650 BIOSIS
 DN PREV200200372650
 TI In vivo vascular engineering: Directed migration of smooth muscle cells to limit neointima.
 AU Wong, Arthur H.; Waugh, Jacob M.; Amabile, Philippe G.; Yuksel, Eser; Dake, Michael D. (1)
 CS (1) Cardiovascular Interventional Radiology, 300 Pasteur Dr., H3648, Stanford, CA, 94305: mddake@stanford.edu USA
 SO Tissue Engineering, (April, 2002) Vol. 8, No. 2, pp. 189-199. http://www.liebertpub.com/TEN. print. ISSN: 1076-3279.
 DT Article
 LA English

=> s l8 and vector?

L9 6 L8 AND VECTOR?

=> dup rem l9

PROCESSING COMPLETED FOR L9

L10 4 DUP REM L9 (2 DUPLICATES REMOVED)

=> d his

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 L4 6 S ENDOTHEL? 3(S) REMODEL?
 L5 0 S L1 AND L4
 L6 4 DUP REM L4 (2 DUPLICATES REMOVED)
 L7 0 S REMODEL? 5(S) L1
 L8 542 S L1 AND REMODEL?
 L9 6 S L8 AND VECTOR?
 L10 4 DUP REM L9 (2 DUPLICATES REMOVED)

=> s l10 not l8

L11 4 L10 NOT L6

=> d bib abs 1-

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L11 ANSWER 1 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
 AN 2001:472794 BIOSIS
 DN PREV200100472794
 TI Identification of a critical Sp1 site within the ***endoglin*** promoter and its involvement in the transforming growth factor-beta stimulation.
 AU Botella, Luisa M.; Sanchez-Elsner, Tilman; Rius, Carlos; Corbi, Angel; Bernabeu, Carmelo (1)
 CS (1) Centro de Investigaciones Biologicas, Consejo Superior de Investigaciones Cientificas, Velazquez 144, 28006, Madrid: bernabeu.c@cib.csic.es Spain
 SO Journal of Biological Chemistry, (September 14, 2001) Vol. 276, No. 37, pp. 34486-34494. print. ISSN: 0021-9258.
 DT Article
 LA English
 SL English
 AB ***Endoglin***, a component of the transforming growth factor-beta (TGF-beta) receptor complex expressed on endothelial cells, is involved in cardiovascular morphogenesis and vascular ***remodeling***, as exemplified by the fact that the ***endoglin*** gene is the target for the autosomal dominant disorder known as hereditary hemorrhagic

telangiectasia type 1. Since haploinsufficiency is the underlying mechanism for hereditary hemorrhagic telangiectasia type 1, understanding the regulation of ***endoglin*** gene expression appears to be a crucial step to correct the disease. In this study we have identified an Sp1 site at -37 as a critical element for the basal transcription of the ***endoglin*** TATA-less promoter. Since ***endoglin*** promoter activity is stimulated by TGF-beta and this stimulation is located at the Sp1-containing proximal region, we have investigated the possible involvement of Sp1 in the TGF-beta-mediated induction. Mutation of the Sp1-binding sequence, or addition of the Sp1 inhibitor WP631, abolished both the basal transcription activity and the TGF-beta responsiveness of the ***endoglin*** promoter. Binding of Sp1 and Smad3 to the proximal promoter region -50/-29 was evidenced by electrophoretic mobility shift assays and DNA affinity precipitation studies. Furthermore, synergistic cooperation on the promoter activity between Sp1 and TGF-beta or Smad3 could be demonstrated by co-transfection experiments of reporter promoter constructs. The molecular mechanism underlying this cooperation appears to involve a direct physical interaction between Sp1 and Smad3/Smad4.

L11 ANSWER 2 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2000:237882 BIOSIS
DN PREV200000237882

TI Adenoviral-mediated expression of antisense RNA to basic fibroblast growth factor reduces tangential stress in arterialized vein grafts.
AU Hanna, Abigail K. (1); Duran, Walter N.; Leconte, Isabelle; Fox, Jonathan C.; Neschis, David G.; Hobson, Robert W., II; Golden, Michael A.
CS (1) Section of Vascular Surgery, Yale University School of Medicine, 333 Cedar St, FMB 137, New Haven, CT, 06520-8062 USA
SO Journal of Vascular Surgery, (April, 2000) Vol. 31, No. 4, pp. 770-780.

ISSN: 0741-5214.

DT Article

LA English

SL English

AB Purpose: The purpose of this study was to test whether basic fibroblast growth factor (bFGF) participates in arterialized vein graft ***remodeling***. Methods: Rabbits underwent in vivo gene transfer and carotid interposition vein grafting. Segments of external jugular vein were infected with an adenovirus that expressed antisense bFGF RNA

(Ad.ASbFGF) at 1 X 10¹⁰ PFU/mL to inhibit new synthesis of bFGF by cells in the vein graft wall. Control rabbits were treated with either adenovirus that encoded beta-galactosidase (Ad.lacZ) at 1 X 10¹⁰ PFU/mL or vehicle (phosphate-buffered saline solution (PBS)). At 3 days, 3 grafts per treatment group were harvested for the determination of gene expression of ASbFGF RNA by reverse transcriptase-polymerase chain reaction. Rabbits were killed, and perfusion was fixed 2 months after the grafting. Total wall thickness and lumen circumference of vein grafts and normal arteries were measured in cross sections. Calculated mean tangential stress (+SD) for the ASbFGF-treated group and controls was compared for significance. Grafts were immunohistochemically stained to assess bFGF protein production. Results: Only the grafts infected with the Ad.ASbFGF gene expressed ASbFGF RNA. Grafts that were treated with Ad.ASbFGF displayed lower tangential stress (10.9 ± 2.3 dynes/cm²) than PBS alone (22 ± 2.8 dynes/cm²) or Ad.lacZ-treated controls (20.6 ± 5.4

dynes/cm²; P < .001). Tangential stress in the Ad.ASbFGF group was comparable to a normal carotid artery (13.9 ± 2.1 dynes/cm²). The difference in mean total wall thickness was significant among the 3 treatment groups: Ad.ASbFGF, 164 ± 3.4 μm; Ad.lacZ, 100 ± 3.3 μm; and PBS, 96 ± 3.6 μm; P < .01). Luminal circumference was not different among the groups. The Ad.ASbFGF-treated vein graft wall was composed of thick layers of concentric smooth muscle cells and ***elastin*** fibers in contrast to the sponge-like appearance observed in control arterialized vein grafts. Reduction in bFGF protein was noted only in the Ad.ASbFGF-treated group. Conclusion: Inhibition of bFGF synthesis in vivo with the use of adenoviral gene transfer of antisense RNA to bFGF promotes a vein graft with decreased tangential stress while maintaining the luminal area. The vein graft wall is ***remodeled*** and qualitatively resembles an artery so that wall tangential stress in Ad.ASbFGF and normal artery are not significantly different. The lack of significant difference in lumen circumference among groups suggests that wall thickening in the Ad.ASbFGF grafts is not at the expense of luminal narrowing. Our results suggest that ASbFGF RNA expression may represent an effective strategy in limiting the failure of arterialized venous conduits.

L11 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2000:110959 BIOSIS
DN PREV200000110959

TI Mechanical strain increases type I collagen expression in pulmonary fibroblasts in vitro.
AU Breen, Ellen C. (1)
CS (1) Dept. of Medicine 0623, Univ. of California, San Diego, 9500 Gilman Dr., La Jolla, CA, 92093-0623 USA
SO Journal of Applied Physiology, (Jan., 2000) Vol. 88, No. 1, pp. 203-209.
ISSN: 8750-7587.

DT Article

LA English

SL English

AB Tissue ***remodeling*** is an adaptive response to mechanical tension in the lung. However, the role of pulmonary fibroblasts in this response has not been well characterized. This study investigates the influence of extracellular matrix on the response of fibroblasts to mechanical strain.

Cells were cultured on flexible-bottom surfaces coated with fibronectin, laminin, or ***elastin*** and exposed to strain. Under these conditions, fibroblasts align perpendicular to the force ***vector***. This stimulus results in an increase in alpha1(I) procollagen mRNA in cells cultured on laminin or ***elastin*** but not fibronectin. Increased alpha1(I) procollagen mRNA was detected 6 h after exposure to strain and reached control levels by 72 h. (3H)proline incorporation into newly synthesized procollagen reflects changes in mRNA levels. Strained fibroblasts cultured on laminin or ***elastin*** incorporated 190 and 114%, respectively, more (3H)proline into procollagen than did unstrained cells. No difference was detected in strained fibroblasts cultured on fibronectin. These results suggest that fibroblasts respond to mechanical strain in vitro, and this response is signaled by cell-extracellular matrix interactions.

L11 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2002 ACS
AN 2002:493975 CAPLUS
DN 137:52335
TI Manipulation of arterial-venous identity
IN Li, Dean Y.
PA The University of Utah Research Foundation, USA

SO PCT Int. Appl., 39 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO. KIND DATE APPLICATION NO. DATE

PI WO 2002011785 A2 20020214 WO 2001-US24405
20010803

WO 2002011785 A3 20020530

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ,
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IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM,
GA, GN,

GQ, GW, ML, MR, NE, SN, TD, TG

US 2002081284 A1 20020627 US 2001-921771
20010803

PRAI US 2000-222759P P 20000803

AB Methods and compns. for manipulating the arterial-venous
identity of

endothelial cells are provided. The methods comprise

introducing an
arterial mol. program into endothelial cells of a vein section such
that

the endothelial cells can ***remodel*** to form arterial
endothelial

cells. The arterial mol. program can comprise one or more
polynucleotides

encoding various genes that are assocd. with arterial
development and/or

differentiation from veins. Expression ***vectors*** comprising
the

genes can be used to introduce the mol. program into the cells.

A method
of treating a patient having an obstructed blood vessel is also
provided.

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